bedfast and all of the supine children, expulsion of infectious material and viable bacteria was largely in an upward direction, and the time of exposure to the glycol vapor was probably considerably longer, in general, than in other situations.

Finally, it is important that during the experimental periods in any ward, glycol vapor was present in the air supply of the subjects during all of the time spent indoors.

These favorable circumstances are, however, largely of quantitative significance. Under other conditions, a higher concentration of glycol in the air or controls on sudden shifts of masses of air into the ward or room might be necessary. The studies described here have demonstrated that the disinfection of air by glycol vapors can be successfully applied to the problem of prevention of air-borne cross-infection.

Summary. The disinfection of air by the vapor of propylene glycol and tri-ethylene glycol has been applied to the clinical problem of the transmission of air-borne cross-infection in a home for convalescent patients. A marked decrease was observed in the total rate of incidence of upper respiratory infections among those patients whose air-supply was largely disinfected by glycol vapor.

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8. EXPERIMENTAL AIR-BORNE TUBERCULOSIS*

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Air-borne Contagion of Tuberculosis in an Animal Room. If normal guinea pigs[1] are placed in individual cages in a room housing tuberculous animals, the exposed pigs acquire tuberculosis of respiratory origin as evidenced by pulmonary lesions, massive tuberculous involvement of the tracheobronchial nodes draining the pulmonary portal of entry and the absence of any disease in the mesenteric or cervical nodes draining the alimentary portal. As is well known, tuberculosis characteristically leaves the traces of its progression in the body by the involvement of the nodes draining the portal of entry.

As may be seen in Figure 1, guinea pigs situated at a considerable distance from their tuberculous room-mates acquire tuberculosis just as often as the guinea pigs placed in immediate proximity to tuberculous animals. Therefore, we may assume that the contagion is uniformly distributed in the room and, since the disease is respiratory in origin, the contagion is air-borne.

Method of Studying Air-borne Contagion of Tuberculosis in Inbred Rabbits of Varying Resistance to the Disease. A large manifold[2] is

* Aided by a grant from the Commonwealth Fund.

separated in the middle by a fine wire mesh screen (Fig. 2). On one side of the screen there is a run for artificially infected rabbits which shed tubercle bacilli in their urine. On the other side of the screen,

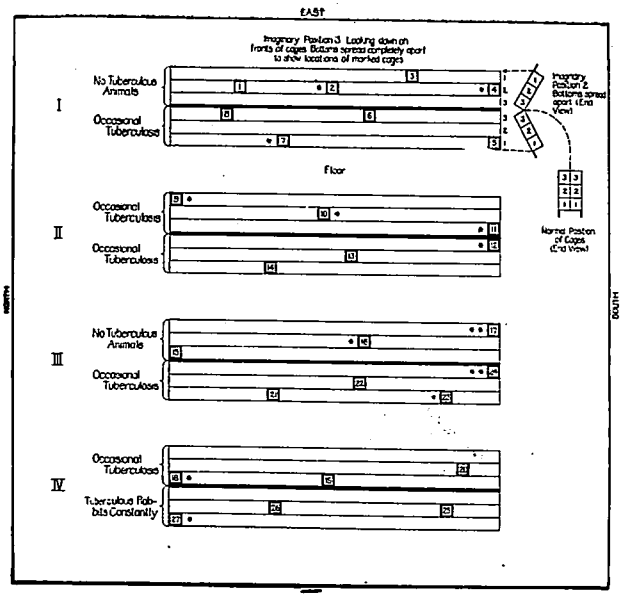

FIG. 1.—Plan of location of cages of tuberculous and non-tuberculous animals. Each asterisk indicates a guinea pig which acquired tuberculosis.

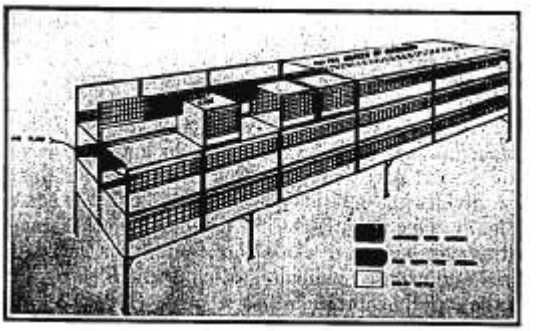

FIG. 2.—Special cages for the study of air-borne tuberculosis in rabbits.

in individual cages with open wire meshwork in back and in front, are placed members of highly inbred rabbit families. By means of tuberculin tests and Roentgen rays the onset of tuberculosis is determined. The disease thus acquired is of respiratory origin and begins as a single

primary focus in the lung, which is always associated, in animals of low genetic resistance (Fig. 3) with a massive tuberculous involvement of the draining tracheobronchial nodes and extensive hematogenous generalization. In the resistant animals (Fig. 4) the disease tends to remain localized to the portal of entry without involvement of the draining nodes and little hematogenous generalization. The disease acquired by these families closely corresponds to the different types of tuberculosis seen in man.

FIG. 3.—Organs of susceptible rabbit F4-33. Duration of disease, 3.3 months. Large, completely caseous primary focus in middle of left lung without softening. Extensive enlargement and massive caseation of homolateral draining tracheobronchial nodes. Corresponding structures on right side not involved. Large nodular caseous tubercles of hematogenous origin in both lungs. Primary, rapidly progressive, generalized tuberculosis with extensive disease in the kidneys, pleura and knee joint.

The Prevention of Natural Air-borne Contagion of Tuberculosis in Rabbits by Ultraviolet Radiation. The room housing the manifold is divided by a solid partition[3] extending from the floor to the ceiling, which also divides the interior of the manifold into 2 equal air-tight halves (Fig. 5). One room is not irradiated. The other room is irradiated. Ultraviolet lamps are placed horizontally in the space between the infected and exposed animals in each of the 3 tiers. In addition, the air of the experimental room as a whole is irradiated by ultraviolet lamps placed above and below this section of the manifold. Littermates of highly inbred families of high and low resistance are placed in corresponding positions in the contact cages of both rooms. The infected rabbits serving as sources of contagion are interchanged daily between the two rooms. Thus both the host and parasite variables are equalized.

FIG. 4.—Organs of resistant rabbit A2-6. Duration of disease, 9 months. Single, large, well-encapsulated cavity in the right lung. Draining tracheobronchial nodes normal. Tuberculosis analogous to the "reinfection type," with tubular spread to the entodermal tract, including larynx and intestines, with ulceration in the colon. Mesenteric nodes normal. Kidneys normal. Non-progressive, uniformly distributed, nodular tubercles in both lungs of hematogenous origin.

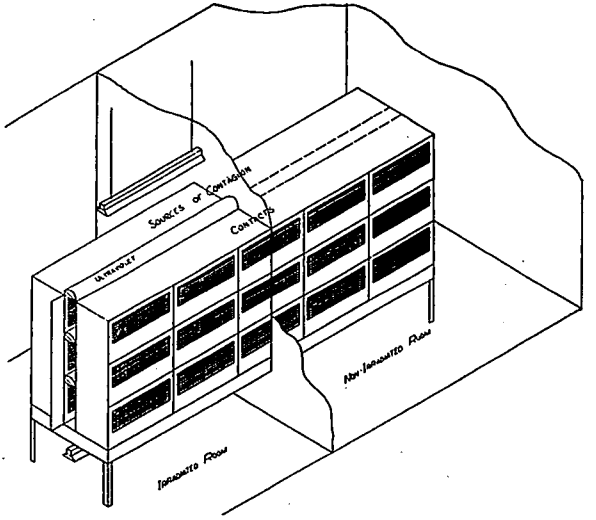

FIG. 5.—Set-up of cages of irradiated and non-irradiated animals.

At the end of a year (Table 1) 11 of the 15 contacts in the unirradiated room died of tuberculosis. These included rabbits of high and low resistance to the disease. None of the 15 litter-mates exposed to the same contagion in the irradiated room developed tuberculosis. Three additional rabbits in the control room developed tuberculin sensitivity without any tuberculous changes at autopsy. None of the protected rabbits developed tuberculin sensitivity. Only one rabbit in the irradiated room developed a regressive microscopic tubercle. Thus a 73% mortality from tuberculosis was eliminated by ultraviolet radiation. That the intensity of contagion involved in this experiment is much greater than occurs in human life can be appreciated from the fact that only 1% of human beings exposed to household contagion acquire tuberculosis in 1 year. It is probable, therefore, that ultraviolet radiation may control air-borne contagion of human tuberculosis.

TABLE 1.—THE EFFECT OF ULTRAVIOLET IRRADIATION ON NATURAL AIR-BORNE CONTAGION OF TUBERCULOSIS IN CONTACTS OF RABBIT FAMILY A, OF HIGH INHERITED NATURAL RESISTANCE, AND IN FAMILIES C AND F, OF LOW INHERITED NATURAL RESISTANCE TO THE DISEASE (EXPERIMENT OF 1942-43)

Family	Rabbit No.	Duration of exposure (mos.)	No. of times tuberculin positive	Maximum tuberculin reaction in mm.² of inflammation	Killed (K) or died (D)	Extent of tuberculosis at death
		Rabbits Exposed in Unirradiated Room				
A	A8=19	5.6	7	368	D	++++
	A8=51	6.3	7	741	D	+++
	A7=26	7.3	7	372	D	++++
	A8=43	9.7	4	475	D	0
	A7=31	10.8	15	1350	D	0
	A8=29	11.6	7	150	K	++
	A7=36	11.6	11	260	K	++
	A7=44	11.6	0	...	K	0
C	C6-9	5.2	7	1330	D	++++
	C6-32	5.7	7	432	D	++++
	C5-50	6.1	7	672	D	++++
	C6-26	9.2	2	213	D	+±
	C6-1	11.6	3	153	K	0
F	F6-25	8.8	4	342	D	++++
	F6-14	10.4	3	141	D	++++
		Rabbits Exposed in Irradiated Room				
A	A8=23	6.5	0	–	D	0
	A8=39	8.9	0	–	D	0
	A7=37	11.6	0	–	K	0
	A7=40	11.6	0	–	K	?*
	A8=49	11.7	0	–	K	0
	A7=28	11.7	0	–	K	0
	A8=52	11.8	0	–	K	0
C	C6-30	5.7	0	–	D	0
	C5-44	11.6	0	–	K	0
	C6-2	11.7	0	–	K	0
	C6-13	11.7	0	–	K	0
	C6-31	11.7	0	–	K	0
F	F5-27	11.7	0	–	K	0
	F6-21	11.8	0	–	K	0
	F6-15	11.8	0	–	K	

* Guinea pig inoculation of a questionable pulmonary lesion demonstrated living, virulent tubercle bacilli.

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Resistance to Attack by Air-borne Tubercle Bacilli and Resistance to the Progress of the Ensuing Disease. One inbred family has little resistance to attack by tuberculosis[4] as indicated by the short interval between the beginning of exposure and the acquisition of a positive tuberculin reaction. Yet it succumbs to a slowly progressive, localizing disease which is of considerable duration, indicating considerable resistance to the progression of the naturally acquired disease. See Table 2.

TABLE 2.—THE RELATION BETWEEN THE RAPIDITY OF ATTACK BY NATURAL AIR-BORNE CONTAGION OF TUBERCULOSIS AND THE DURATION OF THE ACQUIRED DISEASE IN FAMILY A, OF HIGH RESISTANCE, AND FAMILIES F AND C, OF LOW RESISTANCE TO THE INFECTION

A family			F family			C family		
Rabbit No.	Pre-allergic period (mos.)	Duration of disease (mos.)	Rabbit No.	Pre-allergic period (mos.)	Duration of disease (mos.)	Rabbit No.	Pre-allergic period (mos.)	Duration of disease (mos.)
A8=19	0.9	4.7	F4-25	1.9	3.8	C6-9	1.3	3.9
A8=29	1.3	10.4	F4-30	2.4	3.0	C2-8	2.0	9.5
A7=31	1.8	—	F2-15	3.0	6.2	C6-32	2.1	3.6
A8=43	1.8	—	F5-2	4.6	—	C5-50	2.3	3.9
A4-4	2.0	—	F6-25	5.1	3.7	C4R-2	2.7	4.3
A8=51	2.3	4.1	F2-3	6.0	5.0	C5-30	3.0	3.3
A7=26	2.3	5.0	F5-14	6.0	—	C4S-9	3.3	tbc. + snuffles
A3=3	3.2	11.5	F4-33	6.2	3.0	C2-6	3.7	3.7
A6=21	3.3	—	F3-9	6.5	8.0	C4S-11	3.8	tbc. + snuffles
A5=3	3.3	12.9	F6-14	8.3	2.1	C4R-6	4.7	3.8
A5=4	3.3	—	F3-7	11.0	3.5	C4S-30	4.9	—
A5=21	3.3	10.7	F4-11	11.7	4.0	C2-1	5.0	3.0
A7=5	3.5	—				C6-1	5.5	—
A7=36	3.6	8.0				C2-7	7.0	5.0
A5=2	4.3	—				C6-26	8.3	tbc. + snuffles
A7=10	4.9	—				C5-14	9.0	2.6
A2=11	5.0	7.5				C2-18	11.9	—
A7=4	6.0	—						
Average:	3.1±1.3	8.3±3.1		6.1±3.0	4.2±1.7		4.7±2.8	4.2±1.8

P of pre-allergic period between A and F = 0.999.
P of duration of disease between A and F = 0.999.
P of pre-allergic period between A and C = 0.976.
P of duration of disease between A and C = 0.999.

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Another inbred family has considerable resistance against attack by natural air-borne contagion of tuberculosis as indicated by the long interval elapsing between the beginning of exposure and the onset of a positive tuberculin reaction, but the acquired disease is rapidly progressive, disseminating in type and of short duration, indicating little resistance to the progression of the naturally acquired disease. Resistance to attack by air-borne contagion of tuberculosis is therefore distinct from the resistance to the progression of the ensuing disease. "Anfälligkeit" is distinct from "Hinfälligkeit."

The Effect of Increasing Concentrations of Air-borne Tubercle Bacilli on Rabbits of High and Low Genetic Resistance to the Disease. If rabbits of high and low genetic resistance to the disease are simultaneously exposed in different experiments to increasing concentrations of air-borne tubercle bacilli,[4] the effect of this increment is different on animals of high and low resistance. The concentration of air-borne tubercle bacilli is varied by the route of infection used to inoculate the

rabbits serving as sources of contagion. Intravenous inoculation yields fewer rabbits which shed tubercle bacilli in their urine than when the rabbits are inoculated directly in the kidney. The concentration of the infectious agent can also be reduced by using peanut shells instead of peat moss as bedding for the sources of contagion. The former do not adsorb the tubercle bacilli-laden urine as well as the latter, and hence reduce the number of tubercle bacilli-laden particles thrown up in the air by the sources of contagion. The concentration of tubercle bacilli in the air can also be reduced by introducing ultraviolet lights in the room. It was found that increasing concentrations of tubercle bacilli in the environment of the rabbits of high genetic resistance to the disease increase the incidence of the infection, accelerate the rapidity of attack, and affect the essential character of the disease in proportion to the concentration of the infectious agent.

In the rabbits of low genetic resistance to the disease, up to a certain level, increasing concentrations of the infectious agent also increase the incidence of the disease and accelerate its onset. The character of the disease, however, is not affected. It remains rapidly progressive and disseminating in type. Beyond this concentration further increment of the infectious agent has no effect on the incidence of the disease, the rapidity of attack or the character of the ensuing disease.

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9. THE PRESENT STATUS OF GLYCOL VAPORS IN AIR STERILIZATION

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AND

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The practical effectiveness of glycol vapors in the control of infections acquired *via* the respiratory tract depends primarily upon how large a proportion of these infections is air-borne. In man, gradually accumulating evidence indicates that air-borne transmission of such infections as measles,[31] chickenpox,[2] streptococcal infection,[1,6,7,9,18,22,23] the "common cold,"[8,12,27,30] and other infections[13,19] is sufficiently important to encourage practical attempts at air sterilization. Such control may assume the form of a direct attack upon microörganisms